chromosome complement will be found in half of these spores. Thus about one-eighth of the total spores would be deficient for this small segment. Pollen has been examined from only one plant and all of it appeared to be morphologically perfect. Its germinability, however, is not known. It may be that the small deficiency indicated in the hypothesis here outlined would not be lethal to the microgametophyte. Since the plants are propagated vegetatively, these chromosome complexes may be carried on indefinitely.

[1] The author wishes to express his appreciation to Dr. C. E. Allen for valuable suggestions and criticisms.

[2] Research Associate in Botany under a grant from the Wisconsin Alumni Research Foundation.

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ABSENCE OF THE CORPUS CALLOSUM AS A MENDELIZING CHARACTER IN THE HOUSE MOUSE

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Communicated April 26, 1933

In a previous paper[1] there was reported the discovery of a cerebral variation of the house mouse characterized by complete absence of the corpus callosum which is the neocortical commissure normally present in all mammals above the Monotremes. Although a survey of numerous domestic mouse stocks has been made, the variation has been found only among descendants of a cross between rodless and silver strains, designed to determine the strength of linkage existing between the genes determining these two characters. Two cages yielded only normals, nine contained both normals and abnormals while samples from the remaining four cages consisted solely of animals lacking the corpus callosum. Enough data were available to show that the character was not sex-linked and that it was not another manifestation of the rodless gene itself.

Because no behavioristic peculiarity has been found correlated with absence of the corpus callosum, diagnoses of the character have been made by examination of individual brain sections. The necessity of sacrificing each individual diagnosed has rendered the genetic investigation laborious

but in spite of this handicap, sufficient data have been assembled from controlled matings to make certain the mode of its inheritance.

Individuals (usually males) were selected from cages shown to have contained animals lacking the corpus callosum. These were mated to individuals of normal unrelated strains. After the hybrid litters had been raised to weaning age the parents were killed and diagnosed, as were samples of their offspring. From matings of abnormal mice with normals there resulted 54 normal F_1 offspring, which proves the exceptional brain organization to be recessive. From matings in which the parent from abnormal stock died and was not diagnosed, there resulted 21 normals. In this latter mating the undiagnosed parent might have carried two, one or no genes for absent corpus callosum. Yet it is certain, as we shall see that some of these 21 normal progeny carried as a recessive the gene for absent corpus callosum.

Certain heterozygous individuals resulting from these outcrosses were mated *inter se* and produced an F_2 population of 36 animals, 28 of which were normals and 8 of which lacked the corpus callosum. Outcrosses in which the parent from abnormal-producing stock died without diagnosis, yielded in the F_2 13 normals and 4 abnormals. A few matings in which the P_1 parent was normal, but heterozygous for the abnormal gene, produced abnormals in the F_2. This population consisted of 18 normals and 4 abnormals, giving an F_2 total of 59 normals and 16 abnormals where the expectation for a recessive unit-character is 56.3 normals and 18.7 abnormals.

Several matings were made between mice from families known to have produced individuals lacking the corpus callosum. The results of these matings may be readily classified and are found to be fully in accord with the hypothesis that the aberrant character represents the developmental manifestation of a single recessive gene.

Abnormals bred *inter se* are recorded as having produced 1 normal and 10 abnormals. The exceptional normal should not have appeared, if the abnormality is a recessive character and we prefer to explain its occurrence as due to the accidental transfer of an individual from another cage or as a case of faulty diagnosis, rather than as a genetic irregularity. Five other matings in which both parents lacked diagnosis, produced a total of 29 abnormals and no normals.

Where both classes of young appeared as a result of mating abnormals with normals, there were recorded 20 normals and 21 abnormals. Equality is expected upon the basis of a single recessive gene in such back-cross matings. In matings in which only one class appeared there were produced 7 normals.

Where both classes resulted from the mating of abnormals with undiagnosed individuals (doubtless normal), there were recorded 13 normals

and 21 abnormals. Where only one class was produced there were found 7 abnormals, the undiagnosed parent presumably in this case having been abnormal.

When normals were mated to normals there resulted 12 normals. When normals were mated to undetermined individuals there were produced 12 normals and 20 abnormals. The results of these matings are summarized in table 1, in which it will be seen that F_1 outcrosses produced

TABLE 1

TYPE OF MATING	PARENTS	OFFSPRING NORMAL	OFFSPRING ABNORMAL
F_1 Outcross	Abnormal × normal	82	0
F_2 Outcross	Normal (heterozygous) × normal (heterozygous)	59	16
Backcross	Abnormal × normal (heterozygous)	45	62
Inter se	Abnormal × abnormal	1*	46

82 normals, F_2 outcrosses produced 59 normals : 16 abnormals, backcrosses produced 45 normals : 62 abnormals and *inter se* matings produced 46 abnormals and the one exceptional normal (starred) previously explained in the text.

The results of none of these matings are contrary to the interpretation that absent corpus collosum is due to the expression of a single recessive mendelizing gene, which so far as we have been able to observe, confers no detriment upon the individual possessing it. Studies are being made to test the linkage relationships of the gene for absent corpus callosum with the other unit-characters of the house mouse.

[1] King, L. S., and Keeler, C. E., *Proc. Nat. Acad. Sci.*, **18**, 8, 525–528 (1932).